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December 14, 2015

BY E-MAIL AND REGULAR U.S. MAIL
Honorable Mayor David B. Borge
Municipal Building
24 Main Street
Hoosick Falls, NY 12090

Lloyd Wilson
New York State Department of Health
Bureau of Water Supply Protection
Corning Tower,
Room 1110
Empire State Plaza
Albany, NY 12237

Re: Village of Hoosick Falls, New York, PFOA Water Contamination Information

Hon. Mayor Borge and Mr. Wilson:

Over the last 15 years, I have represented tens of thousands of individuals in various communities across the country who have been injured because of the contamination of their drinking water with PFOA. As a result of a 2005 settlement of the claims of approximately 70,000 such individuals exposed to PFOA-contaminated drinking water in Ohio and West Virginia, in which I served as class counsel for the residents, extensive investigation and study into the actual human health effects of community-level exposure to PFOA in drinking water was undertaken by an independent panel of scientists known as the "C8 Science Panel." Under the terms of that class action settlement, the C8 Science Panel spent over seven years and over $30 Million analyzing data from approximately 69,000 residents (including actual PFOA blood results from tens of thousands of such residents and decades of actual medical records and actual drinking water exposure histories) to determine which diseases

actually were linked to community resident exposures to PFOA in drinking water for as little as one year and at PFOA concentrations as low as 0.05 ppb.[1]

By the end of 2012, the C8 Science Panel had determined, after reviewing all of the data from the tens of thousands of community residents and all available scientific data (epidemiological and toxicological), that human exposure to PFOA in that community, which included individuals exposed for as little as one year to drinking water with as low as 0.05 ppb PFOA, had a probable link to six serious diseases: 1) kidney cancer; 2) testicular cancer; 3) ulcerative colitis; 4) thyroid disease; 5) preeclampsia/pregnancy-induced hypertension; and 6) medically-diagnosed high cholesterol (hypercholesterolemia). The C8 Science Panel's final reports on each of these diseases (and links to the peer-reviewed, published papers confirming all of their findings and the scientific standards they used) are available through the C8 Science Panel's website: www.c8sciencepanel.org.

As a result of these independent scientific findings from the C8 Science Panel, the corporation responsible for contaminating the residents' water with PFOA, E. I. du Pont de Nemours and Company ("DuPont"), agreed not to contest the fact that PFOA is capable of causing each of these six serious diseases among the residents so-exposed. As a result, individuals in those communities who were exposed to at least 0.05 ppb PFOA in their drinking water for more than one year and who contracted (or died from) one or more such diseases are now pursuing personal injury, wrongful death, and punitive damage claims against DuPont for damages. The first of approximately 3500 such cases now pending against DuPont went to trial in an Ohio federal court this September, resulting in a $1.6 Million jury verdict in favor of a community resident who contracted kidney cancer after being exposed for several years to PFOA in her drinking water at concentrations between approximately 0.17 – 0.47 ppb.

It is in this context that we write to address certain statements that are being made to the community in Hoosick Falls, New York, with respect to the potential health risks posed by the level of PFOA that we recently saw reported to be as high as 0.6 ppb in that community's drinking water. In particular, we agree with the position taken by US EPA's Region 2 in its November 2, 2015, letter to the Village of Hoosick Falls that the Village (and New York Department of Health ("DOH")) should make clear in any information provided to residents (on websites, in public meetings, or otherwise) that the level of PFOA found in the Village's drinking water supply exceeds the 0.4 ppb provisional health advisory level that US EPA established for *short-term* (weeks to months – not years) exposure to PFOA in drinking water, mandating immediate alternate drinking water sources for the community.[2] We further agree that steps should

[1] More information on that settlement is available at: www.C8claim.com.
[2] As noted by US EPA Region 2 in its November 25, 2015, letter to the Village, US EPA has not yet released a guideline for long-term (more than several weeks or months) exposure to PFOA in drinking water. We have been asking US EPA to move forward in

be taken to make sure that the drinking water is treated or filtered for PFOA before being supplied to residents.

These levels of PFOA in the Village's drinking water present a health concern, not only because they exceed the current short-term EPA drinking water advisory, but because they represent levels of PFOA that are significantly above the threshold levels of PFOA exposure where the C8 Science Panel found links with six very serious diseases, including two forms of cancer. As noted above, the community exposures to PFOA studied by the C8 Science Panel where the six serious disease links were found included residents exposed to as little as 0.05 ppb in their drinking water for as little as one year. The 0.6 ppb level recently reported to be present in the Hoosick Falls drinking water is ***12 times higher*** than this minimum exposure level for the C8 Science Panel study population. This fact calls into question the statement made in DOH's December 2015 "Long Fact Sheet" provided to the Village (and posted on the Village's website for its residents) that it does "not expect health effects to occur" from continued use of the Village's drinking water with 0.6 ppb PFOA. In this regard, we encourage the Village and DOH to review the C8 Science Panel's work and to make links to that work available through the Village's and Agency's websites so that the community has ready access to as much scientific information as possible with respect to human health effects and risks attributable to their particular community-level exposures to PFOA in drinking water.[3]

We would be happy to discuss any of this publicly-available data with you in further detail. Thank you.

Sincerely yours,

Robert A. Bilott

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that regard for well over a decade. The limit for such long-term exposures likely would be significantly lower than the 0.4 ppb guideline for only short-term, acute exposures, as evidenced by the 0.04 ppb guideline (10 tens lower than the EPA's short-term guideline) recommended by the State of New Jersey back in 2007 (even before the C8 Science Panel's findings on serious disease links were released) for long-term exposures to PFOA in drinking water. (*See* Ex. 1.)

[3] We also note that the Village appears to have a link to the 2012 EPA "Fact Sheet" for PFOA on its website, instead of the more-current 2014 "Fact Sheet" (copy enclosed at Ex. 2). This newer Fact Sheet specifically notes that "[s]tudies also indicate that continued exposure to low levels of PFOA in drinking water may result in adverse health effects."

Encls.
cc: Judith A. Enck (US EPA Region 2) (w/encls.)
Nathan Graber (NYSDOH) (w/encls.)

EXHIBIT 1

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January 20, 2015

BY EMAIL AND REGULAR U.S. MAIL

Susan Hedman
Regional Administrator
United States Environmental Protection Agency
Region V
77 West Jackson Blvd.
Mail Code: R-19J
Chicago, IL 60604-3507

Shawn M. Garvin
Regional Administrator
United States Environmental Protection Agency
Region III
1650 Arch Street
Mail Code: 3RA00
Philadelphia, PA 19103-2029

Randy C. Huffman
Cabinet Secretary
West Virginia Department of Environmental Protection
601 57th Street, SE
Charleston, WV 25304

Re: *In the Matter of: E.I. du Pont de Nemours and Company*
(Docket Nos. SDWA-03-2009-0127 DS – SDWA-05-2009-0001)

Dear Ms. Hedman, Mr. Garvin and Mr. Huffman:

We first wrote to US EPA and WVDEP in March of 2001 – over 13 years ago – to alert your Agencies to the imminent and substantial threat to human health and the environment posed by the contamination of human drinking water supplies with perfluorooctanoic acid ("PFOA" a/k/a "C-8") released from E. I. du Pont de Nemours and Company's ("DuPont's) Washington Works Plant in Wood County, West Virginia (the "DuPont Plant"). (*See* Ex. A.) In that original letter, we alerted your Agencies to the fact that PFOA was poisoning drinking water supplies in the vicinity of the DuPont Plant at levels exceeding a 1 part per billion (1 ppb) exposure guideline that DuPont had adopted for PFOA in community water more than a decade earlier, and asked your

Agencies to take immediate action to address and abate that health threat under applicable state and federal laws, including the Clean Water Act ("CWA"), the Safe Drinking Water Act ("SDWA"), the Toxic Substances Control Act ("TSCA"), and the Resource Conservation and Recovery Act ("RCRA"). (*See id.*) Soon thereafter, US EPA launched a "priority review" of PFOA under TSCA and began the process to establish federal safety limits for PFOA in drinking water, beginning with the release of a draft PFOA risk assessment in 2003. WVDEP, on the other hand, has still not even begun the process of trying to establish or set any regulatory safety levels for PFOA, choosing, instead, to defer to whatever US EPA ultimately decides. In the meantime, given the lack of any enforceable federal or state regulatory safety limits for PFOA in drinking water, US EPA was left with having to address this serious health threat by negotiating "Consent Orders" with DuPont through which US EPA could incorporate only such terms as to which DuPont ultimately would "consent."

The first such US EPA Consent Order was entered in 2002, soon after US EPA received our original letter. Rather than require clean water whenever DuPont's own 1 ppb drinking water exposure level was exceeded (which 1 ppb level had been created by DuPont's own scientists, had been followed internally by DuPont for more than a decade, and was still being followed internally by DuPont at that time), DuPont would only "consent" to providing clean water through this new Consent Order, if the level of PFOA exceeded a significantly higher 14 ppb level that DuPont's outside consultants had generated.

Just two months later, in May 2002, DuPont succeeded in forcing US EPA to raise that 14 ppb level to 150 ppb, based on the terms of a separate, privately-negotiated deal between DuPont and WVDEP under which WVDEP allowed DuPont to collaborate with WVDEP and its consultant to create a new, higher trigger level for clean water. DuPont then held that 150 ppb number out to the public for the next several years as the appropriate, government-endorsed safety number for PFOA in drinking water, even though, internally, DuPont's own scientists still supported a 1 ppb exposure guideline for PFOA in community drinking water supplies.

DuPont only "consented" to a new Consent Order with US EPA on these issues in 2006, after significant additional health risk information had been released on PFOA, including a final report from US EPA's own Science Advisory Board, where the majority of the Board recommended that PFOA be classified as a "likely" human carcinogen. Upon review of this new data, US EPA's scientists had determined that the 150 ppb trigger picked by DuPont and WVDEP was "not protective of human health and must be replaced by a lower threshold value of 0.20 ppb." (Ex. B. at 1.) DuPont informed US EPA at the time that it agreed, based on this new data, that "it is prudent to minimize, where possible, exposure to biopersistent materials such as PFOA," and that a new, lower clean water trigger number should be adopted "to help promote reductions of PFOA in blood levels through alternate drinking supplies." (Ex. C at 3-4.) According to DuPont, a "median serum/drinking water ratio for PFOA was calculated to be 105, i.e.,

for every 1 ppb of PFOA in drinking water ingested by community residents; 105 ppb of PFOA will be present in serum." (*Id.* at 9.) At the 150 ppb trigger level then in effect, DuPont noted that "a serum level of approximately 15 ppm [15,000 ppb] can be predicted," which "exceeds the current occupational exposures" where adverse health effects were being reported in the new data. (*Id.* at 11.) According to DuPont, reducing the clean water trigger from 150 ppb to 0.5 ppb - not 0.20 ppb – would be sufficient, as it "would result in approximately 50 ppb of PFOA in serum," which DuPont argued was "within the range found in the general population" where no such adverse health effects were purportedly being found at the time. (*Id.*) Thus, in light of DuPont's refusal to agree to a safe drinking water trigger level any lower than 0.5 ppb at that time, the new US EPA/DuPont Consent Order in 2006 lowered the PFOA clean drinking water threshold from 150 ppb to 0.5 ppb PFOA. US EPA was not able to obtain DuPont's "consent" to lower the threshold for safe water any further until 2009, after US EPA released its first "provisional health advisory" ("PHA") for short-term, temporary exposure to C-8 in drinking water of 0.4 ppb. At that point, DuPont finally agreed to lower the clean water trigger in its Consent Order with US EPA - but only from 0.5 ppb to 0.4 ppb.

US EPA made clear in its 2009 Consent Order with DuPont that the 0.4 ppb C-8 trigger level for clean water was a "temporary value that will be re-evaluated when EPA determines a reference dose under TSCA or establishes a drinking water standard for C-8, whichever comes first." (2009 Consent Order, at ¶ 46.) US EPA also made clear that it reserved "the right to modify the [0.4 ppb C-8 clean water trigger] identified in this Order if information previously unknown to EPA is received and EPA determines that this previously unknown information, together with any other relevant information, indicates that [such trigger level] may not be protective of human health." (*Id.* at ¶ 47.)

Since entry of the current Consent Order in March of 2009, extensive additional information has been released in the scientific and peer-reviewed literature confirming that the 0.4 ppb trigger level for clean water is not protective of human health for long-term exposures and should be revised. For example, in December 2009, US EPA released its Long-Chain Perfluorinated Chemicals (PFCs) Action Plan, identifying C-8 as "raising serious health and environmental concerns," which could justify significant "risk reduction measures to protect human health and the environment." Then, in 2011-2012, an independent C-8 Science Panel – jointly selected and fully-funded by DuPont – confirmed probable links between exposure to PFOA in drinking water as low as 0.05 ppb and six serious human diseases: 1) kidney cancer; 2) testicular cancer; 3) ulcerative colitis; 4) thyroid disease; 5) pregnancy-induced hypertension/preeclampsia; and 6) hypercholesterolemia. Each of those links was based on the independent Science Panel's review of data (including PFOA blood tests, blood chemistries, and medical records reviews/verifications) from approximately 70,000 people actually exposed to PFOA in drinking water in the vicinity of the DuPont Plant, along with all other available data, including peer-reviewed studies from all over the world and DuPont's own worker data. Each of the Science Panel's findings ultimately was

confirmed in published, peer-reviewed papers. US EPA was encouraged through public comments and formal peer reviewers to consider and incorporate all such important new data (along with additional, significant new toxicological data, including new data on mammary gland impacts and from studies in mice), in the context of finalizing US EPA's "Health Effects Document for Perfluorooctanoic Acid," which was released in draft form to the public in 2014 but, as of today's date, still has not been finalized.

Although US EPA still has not released a guideline for long-term, chronic exposure to PFOA in drinking water or finalized its PFOA health effects document, European regulators have moved forward. Just this month, the European Chemicals Agency (ECHA) publicly released a report from Germany and Norway recommending significant new restrictions on PFOA in light of the more current health effects data, specifically including the findings of the C8 Science Panel linking very low level PFOA exposure in drinking water (as low as 0.05 ppb) with 6 diseases, including two forms of cancer. (*See* http://echa.europa.eu/documents/10162/e9cddec6-3164-473d-b590-8f9caa50e7 .) Particularly significant in this new European report are new risk calculations revealing that levels of PFOA in the blood of people exposed to PFOA at the levels allowed under the existing 2009 Consent Order (PFOA drinking water levels as high as 0.5 ppb) would far exceed the blood risk levels derived using the latest health effects data. This is because significant adverse health effects (including cancer) were found to be linked to PFOA exposures in humans as low as 0.05 ppb in drinking water – some *ten times lower* than the current level allowed under the 2009 Consent Order. (*See also* Post, G.B., *et al.*, "Perfluorooctanoic acid (PFOA), an emerging drinking water contaminant: A critical review of recent literature," 116 *Environ. Res.* 93-117 (July 2012).)

Although neither the European report nor US EPA's work to set a safety level for long-term chronic exposure to PFOA in drinking water has been completed, US EPA retains both the right and responsibility to modify the 2009 Consent Order in light of new health data on PFOA to make sure that human health is protected. US EPA should consider the new PFOA health effects data and European safety calculations noted above to evaluate whether there is a current or imminent and substantial threat or endangerment to human health that mandates steps be taken to modify the 2009 Consent Order to require DuPont to provide for alternate/clean drinking water for any human drinking water supply in the vicinity of the DuPont Washington Works Plant where PFOA has been detected at levels below the current 0.4 ppb trigger level established in that Consent Order. In New Jersey, for example, state regulators already are evaluating the safety of drinking water supplies by comparing PFOA water levels to a 0.04 ppb "health-based drinking water guidance level" developed specifically for the purpose of assessing long-term, chronic exposures to PFOA in human drinking water supplies. (*See, e.g.,* Ex. D.)

As both US EPA and WVDEP are aware, there are at least two public drinking water supplies in the vicinity of the DuPont Plant in West Virginia where sampling for

PFOA revealed levels of PFOA in the treated water above the 0.05 ppb level of exposure considered in the C8 Science Panel studies: 1) the City of Parkersburg, West Virginia (most recent rounds of CCL3 sampling data submitted to US EPA and now posted on US EPA's website revealed PFOA as high as 0.0631 ppb after treatment on 3/25/14); and 2) the City of Vienna, West Virginia (reports submitted by DuPont to US EPA and posted in US EPA' public dockets confirm 0.056 ppb PFOA after treatment on last-known PFOA sampling date of 5/10/07). (*See* Ex. E). DuPont successfully used US EPA's and WVDEP's continuing failure to adopt any final long-term, chronic exposure limits for PFOA in drinking water to thwart all efforts by impacted Parkersburg residents to require DuPont to provide clean water through the court system. (*See, e.g.,* 9/30/08 Memorandum Opinion and Order in *Rhodes, et al., v. E.I. du Pont de Nemours and Co.,* Civil Action No. 6:06-cv-00530 (S.D. W. Va.) at 1 (West Virginia federal court denied Parkersburg residents' attempts to bring community/class-wide claims against DuPont for clean water through the judicial system, noting that, although the "plaintiffs have presented compelling evidence that exposure to C-8 may be harmful to human health, and the evidence certainly justifies the concerns expressed by the plaintiffs in this case," the Court could not certify those claims to proceed through the Court system at that time: "The fact that a public health risk may exist is more than enough to raise concern in the community and call government agencies to action, but it does not show the common individual injuries needed to certify a class action" for relief through the judicial system.).)

Thus, despite DuPont's acknowledgment to US EPA by at least 2006 that "it is prudent to minimize, where possible, exposure to biopersistent materials such as PFOA" and purported desire "to help promote reductions in PFOA in blood levels through alternate drinking supplies," (Ex. C at 3-4), DuPont aggressively fought and ultimately succeeded in preventing Parkersburg residents from obtaining clean water through the court system, even though DuPont knew that failure to remove PFOA from that water would allow PFOA to steadily build up and accumulate in the blood of the residents drinking that water at a ratio of approximately 105 ppb PFOA in blood for every 1 ppb PFOA in their drinking water. US EPA and WVDEP, likewise, have not required any action to date to abate these on-going exposures in either Parkersburg or Vienna, despite knowledge of the on-going contamination (and associated accumulation and build- up of PFOA in residents' blood) for almost a decade.

US EPA should re-assess its position with respect to these on-going PFOA exposures in light of existing health data. US EPA also should consider whether any steps need to be taken to insure that the appropriate parties remain bound under its existing Consent Orders and Memoranda of Understanding with DuPont on PFOA issues, in light of DuPont's recently announced intentions to soon "spin-off" and/or jettison certain operations and liabilities of DuPont relating to PFOA to a new entity to be known as "Chemours," (*see* Ex. F).

Very truly yours,

Robert A. Bilott

RAB:mdm
Encls: Exs. A-F
cc: Elizabeth Doyle, USEPA (w/encls.)(by regular U.S. mail)

EXHIBIT 2

United States Environmental Protection Agency

Emerging Contaminants – Perfluorooctane Sulfonate (PFOS) and Perfluorooctanoic Acid (PFOA)

March 2014

EMERGING CONTAMINANTS FACT SHEET – PFOS and PFOA

At a Glance

- Fully fluorinated compounds that are human-made substances and are not naturally found in the environment.
- Used as a surface-active agent and in a variety of products, such as firefighting foams, coating additives and cleaning products.
- Do not hydrolyze, photolyze or biodegrade under typical environmental conditions and are extremely persistent in the environment.
- Studies have shown they have the potential to bioaccumulate and biomagnify in wildlife.
- Readily absorbed after oral exposure and accumulate primarily in the serum, kidney and liver.
- Toxicological studies on animals indicate potential developmental, reproductive and systemic effects.
- Health-based advisories or screening levels for PFOS and PFOA have been developed by the EPA and state agencies.
- Standard detection methods include high-performance liquid chromatography and tandem mass spectrometry.
- Common ex situ water treatment technologies include activated carbon filters and reverse osmosis units.

Introduction

An "emerging contaminant" is a chemical or material that is characterized by a perceived, potential, or real threat to human health or the environment or by a lack of published health standards. A contaminant may also be "emerging" because a new source or a new pathway to humans has been discovered or a new detection method or treatment technology has been developed (DoD 2011). This fact sheet, developed by the U.S. Environmental Protection Agency (EPA) Federal Facilities Restoration and Reuse Office (FFRRO), provides a summary of the emerging contaminants perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA), including physical and chemical properties; environmental and health impacts; existing federal and state guidelines; detection and treatment methods; and additional sources of information. This fact sheet is intended for use by site managers who may address PFOS and PFOA at cleanup sites or in drinking water supplies and for those in a position to consider whether these chemicals should be added to the analytical suite for site investigations.

PFOS and PFOA are extremely persistent in the environment and resistant to typical environmental degradation processes. As a result, they are widely distributed across the higher trophic levels and are found in soil, air and groundwater at sites across the United States. The toxicity, mobility and bioaccumulation potential of PFOS and PFOA pose potential adverse effects for the environment and human health.

What are PFOS and PFOA?

- PFOS and PFOA are fully fluorinated, organic compounds and are the two perfluorinated chemicals (PFCs) that have been produced in the largest amounts within the United States (ATSDR 2009; EFSA 2008).
- PFOS is a perfluoralkyl sulfonate that is commonly used as a simple salt (such as potassium, sodium or ammonium) or is incorporated into larger polymers (EFSA 2008; EPA 2009c).
- PFOA is a perfluoralkyl carboxylate that is produced synthetically as a salt. Ammonium salt is the most widely produced form (EFSA 2008; EPA 2009c).

What are PFOS and PFOA? (continued)

- PFOS synonyms include 1-octanesulfonic acid, heptadecafluoro-, 1-perfluorooctanesulfonic acid, heptadecafluoro-1-octanesulfonic acid, perfluoro-n-octanesulfonic acid, perfluoroctanesulfonic acid and perfluoroctylsulfonic acid (ATSDR 2009; UNEP 2005).
- PFOA synonyms include pentadecafluoro1-octanoic acid, pentadecafluoro-n-octanoic acid, pentadecaflurooctanoic acid, perfluorocaprylic acid, perfluoroctanoic acid, perfluoroheptanecarboxylic acid and octanoic acid (ATSDR 2009).
- They are stable chemicals that include long carbon chains. Because of their unique lipid- and water-repellent characteristics, PFOS and PFOA are used as surface-active agents in various high-temperature applications and as a coating on surfaces that contact with strong acids or bases (Schultz and others 2003; UNEP 2005).
- PFCs are used in a wide variety of industrial and commercial products such as textiles and leather products, metal plating, the photographic industry, photolithography, semi-conductors, paper and packaging, coating additives, cleaning products and pesticides (ATSDR 2009; EPA 2009c; OECD 2002).
- Through 2001, PFCs were used to manufacture Aqueous Film Forming Foam (AFFF). PFOS-based AFFF is used to extinguish flammable liquid fires (for example, hydrocarbon fueled), such as fires involving gas tankers and oil refineries (EPA 2013a; DoD SERDP 2012).
- They are human-made compounds and do not occur naturally in the environment (ATSDR 2009; EPA 2009c).
- PFOS and PFOA can also be formed by environmental microbial degradation or by metabolism in larger organisms from a large group of related substances or precursor compounds (ATSDR 2009; UNEP 2006).
- The 3M Company, the primary manufacturer of PFOS, completed a voluntary phase-out of PFOS production in 2002 (ATSDR 2009; 3M 2008).

Exhibit 1: Physical and Chemical Properties of PFOS and PFOA
(ATSDR 2009; Brooke and others 2004; EFSA 2008; Environment Canada 2012; EPA 2002b; OECD 2002; UNEP 2006)

Property	PFOS (Potassium Salt)	PFOA (Free Acid)
Chemical Abstracts Service (CAS) Number	2795-39-3	335-67-1
Physical Description (physical state at room temperature and atmospheric pressure)	White powder	White powder/ waxy white solid
Molecular weight (g/mol)	538	414
Water solubility at 25°C (mg/L)	550 to 570 (purified), 370 (fresh water), 25 (filtered sea water)	9.5×10^3 (purified)
Melting Point (°C)	> 400	45 to 54
Boiling point (°C)	Not measurable	188 to 192
Vapor pressure at 20 °C (mm Hg)	2.48×10^{-6}	0.017[1]
Octanol-water partition coefficient (log K_{ow})	Not measurable	Not measurable
Organic-carbon partition coefficient (log K_{oc})	2.57 (Value estimated based on anion and not the salt)	2.06
Henry's law constant (atm-m^3/mol)	3.05×10^{-9}	Not measurable
Half-Life	Atmospheric: 114 days Water: > 41 years (at 25° C)	Atmospheric: 90 days[2] Water: > 92 years (at 25° C)

Abbreviations: g/mol – grams per mole; mg/L – milligrams per liter; °C – degree Celsius; mm Hg – millimeters of mercury; atm-m^3/mol – atmosphere-cubic meters per mole.
[1] Extrapolation from measurement.
[2] The atmospheric half-life value identified for PFOA is estimated based on available data determined from short study periods.

What are PFOS and PFOA? (continued)

- PFOS chemicals are no longer manufactured in the United States; however, EPA significant new use rules (SNURs) allow for the continuation of a few, limited, highly technical applications of PFOS-related substances where no known alternatives are available. In addition, existing stocks of PFC-based chemicals that were manufactured or imported into the United States before the effective date of the SNURs (for example, PFOS-based AFFF produced before the rules took effect in 2002) can still be used (EPA 2009c, 2013a).
- PFOA as its ammonium salt is manufactured primarily for use as an aqueous dispersion agent and in the manufacture of fluoropolymers (which are used in a wide variety of mechanical and industrial components) such as electrical wire casings, fire- and chemical-resistant tubing and plumbing seal tape. They are also produced unintentionally by the degradation of some fluorotelomers (ATSDR 2009; EPA 2009c).
- As part of the EPA's PFOA stewardship program, eight companies committed to achieve the following by 2010: (1) reduce global facility emissions of PFOA to all media; (2) reduce precursor chemicals that break down to PFOA and related higher homologue chemicals; and (3) PFOA product content (95 percent). The companies also agreed to work toward eliminating these chemicals from emissions and products by 2015 (EPA 2013a).

What are the environmental impacts of PFOS and PFOA?

- During past manufacturing processes, large amounts of PFOS and PFOA were released to the air, water and soil in and around fluorochemical facilities (ATSDR 2009).
- PFOS and PFOA have been detected in a number of U.S. cities in surface water and sediments downstream of former fluorochemical production facilities and in wastewater treatment plant effluent, sewage sludge and landfill leachate (EPA 2002b; OECD 2002).
- The environmental release of PFOS-based AFFF may also occur from tank and supply line leaks, use of aircraft hangar fire suppression systems and firefighting training (DoD SERDP 2012).
- Both PFOS and PFOA are the stable end products resulting from the degradation of precursor substances through a variety of abiotic and biotic transformation pathways (Conder and others 2010).
- Because of their chemical structure, PFCs, including PFOS and PFOA, are chemically and biologically stable in the environment and resist typical environmental degradation processes, including atmospheric photooxidation, direct photolysis and hydrolysis. As a result, these chemicals are extremely persistent in the environment (OECD 2002; Schultz and others 2003).
- PFOS and PFOA have very low volatility because of their ionic nature. Therefore, they will be persistent in water and soil (3M 2000; ATSDR 2009).
- When released directly to the atmosphere, PFCs are expected to adsorb to particles and settle to the ground through wet or dry deposition (Barton and others 2007; Hurley and others 2004).
- In their anionic forms, PFOA and PFOS are water-soluble and can migrate readily from soil to groundwater, where they can be transported long distances (Davis and others 2007; Post and others 2012).
- Monitoring data from the Arctic region and at sites remote from known point sources have shown levels of PFOS and PFOA in environmental media and biota, indicating that long-range transport has occurred. For example, PFOA and PFOS have been detected in concentrations from the low- to mid- picograms per liter (pg/L) range in remote regions of the Arctic caps. In addition, PFOS concentrations detected in the liver of the Canadian Arctic polar bear range from 1,700 to more than 4,000 nanograms per gram (ng/g) (Lau and others 2007; Martin and others 2004; Young and others 2007).
- Causes of long-range PFC transport include (1) atmospheric transport of precursor compounds (such as perfluoroalkyl sulfonamides), followed by degradation to form PFCs and (2) direct, long-range transport of PFCs via ocean currents or in the form of marine aerosols (Armitage and others 2006; Post and others 2012).

What are the environmental impacts of PFOS and PFOA? (continued)

- The wide distribution of PFCs increases the potential for bioaccumulation and bioconcentration as they are transferred from low to higher trophic level organisms. Because of their persistence and long-term accumulation, higher trophic level wildlife such as fish, piscivorous birds and other biota can continue to be exposed to PFOS and PFOA (EPA 2006a; UNEP 2006).
- The bioaccumulation potential of PFCs increases with increasing carbon chain length (ATSDR 2009; Furdui and others 2007).
- PFOS is the only PFC that has been shown to accumulate to levels of concern in fish tissue. The estimated bioconcentration factor in fish ranges from 1,000 to 4,000 (EFSA 2008; MDH 2011; OECD 2002).
- As of 2013, the Superfund Information Systems Database indicates PFCs have been reported in the 5-year reviews of 14 hazardous waste sites on the EPA National Priorities List (EPA 2013b).
- Data gathered in 2008 from the DoD Knowledge Based Corporate Reporting System show that 594 DoD facilities have been categorized as Fire/Crash/Training Sites and, therefore, have the potential for PFC contamination based on historical use of AFFF (DoD 2008; DoD SERDP 2012).

What are the routes of exposure and the health effects of PFOS and PFOA?

- Studies have found PFOS and PFOA in the blood samples of the general human population and wildlife nationwide, indicating that exposure to the chemicals is widespread (ATSDR 2009; EPA 2006a).
- Reported data indicate that serum concentrations of PFOS and PFOA are higher in workers and individuals living near fluorochemical production facilities than for the general population (Calafat and others 2007; EPA 2009c).
- Potential pathways, which may lead to widespread exposure, include ingestion of food and water, use of commercial products or inhalation from long-range air transport of PFC-containing particulate matter (ATSDR 2009; EPA 2009c).
- Based on the limited information available, fish and fishery products seem to be one of the primary sources of human exposure to PFOS (EFSA 2008).
- While a federal screening level or toxicity value for the consumption of fish has not yet been established, the Dutch National Institute for Public Health and the Environment has calculated a maximum permissible concentration for PFOS of 0.65 nanograms per liter (ng/L) for fresh water (based on consumption of fish by humans as the most critical route) (Moermond and others 2010).
- Studies also indicate that continued exposure to low levels of PFOA in drinking water may result in adverse health effects (Post and others 2012).
- Toxicology studies show that PFOS and PFOA are readily absorbed after oral exposure and accumulate primarily in the serum, kidney and liver. No further metabolism is expected (EPA 2006a, 2009c).
- PFOS and PFOA have half-lives in humans ranging from 2 to 9 years, depending on the study. This half-life results in continued exposure that could increase body burdens to levels that would result in adverse outcomes (ATSDR 2009; EPA 2009c; Kärrman and others 2006; Olsen and others 2007).
- Acute- and intermediate-duration oral studies on rodents have raised concerns about potential developmental, reproductive and other systemic effects of PFOS and PFOA (Austin and others 2003; EPA 2006a).
- The ingestion of PFOA-contaminated water was found to cause adverse effects on mammary gland development in mice (Post and others 2012).
- One study indicated that exposure to PFOS can affect the neuroendocrine system in rats; however, the mechanism by which PFOS affects brain neurotransmitters is still unclear (Austin and others 2003).
- Both PFOS and PFOA have a high affinity for binding to B-lipoproteins and liver fatty acid-binding protein. Several studies on animals have shown that these compounds can interfere with fatty acid metabolism and may deregulate metabolism of lipids and lipoproteins (EFSA 2008; EPA 2009c).

What are the routes of exposure and the health effects of PFOS and PFOA? (continued)

- In May 2006, the EPA Science Advisory Board suggested that PFOA cancer data are consistent with the EPA guidelines for the Carcinogen Risk Assessment descriptor "likely to be carcinogenic to humans." EPA is still evaluating this information and additional research pertaining to the carcinogenicity of PFOA (EPA 2006b, 2013a).
- The American Conference of Governmental Industrial Hygienists (ACGIH) has classified PFOA as a Group A3 carcinogen — confirmed animal carcinogen with unknown relevance to humans (ACGIH 2002).
- The chronic exposure to PFOS and PFOA can lead to the development of tumors in the liver of rats; however, more research is needed to determine if there are similar cancer risks for humans (ATSDR 2009; OECD 2002).
- In a retrospective cohort mortality study of more than 6,000 PFOA-exposed employees at one plant, results identified elevated standardized mortality ratios for kidney cancer and a statistically significant increase in diabetes mortality for male workers. The study noted that additional investigations are needed to confirm these findings (DuPont 2006; Lau and others 2007).
- Studies have shown that PFCs may induce modest effects on reactive oxygen species and deoxyribonucleic acid (DNA) damage in the cells of the human liver (Eriksen and others 2010; Reistad and others 2013).
- Analysis of U.S. National Health and Nutrition Examination Survey representative study samples indicate that higher concentrations of serum PFOA and PFOS are associated with thyroid disease in the U.S. general adult population. Further analysis is needed to identify the mechanisms underlying this association (Melzer and others 2010).
- Epidemiologic studies have shown an association between PFOS exposure and bladder cancer; however, further research and analysis are needed to understand this association (Alexander and others 2004; Lau and others 2007).

Are there any federal and state guidelines and health standards for PFOS and PFOA?

- In January 2009, the EPA's Office of Water established a provisional health advisory (PHA) of 0.2 micrograms per liter (µg/L) for PFOS and 0.4 µg/L for PFOA to assess the potential risk from short-term exposure of these chemicals through drinking water. PHAs reflect reasonable, health-based hazard concentrations above which action should be taken to reduce exposure to unregulated contaminants in drinking water (EPA 2009d, 2013a).
- EPA Region 4 calculated a residential soil screening level of 6 milligrams per kilogram (mg/kg) for PFOS and 16 mg/kg for PFOA (EPA Region 4 2009).
- Various states have established drinking water and groundwater guidelines, including the following:
  - Minnesota has established a chronic health risk limit of 0.3 µg/L for PFOS and PFOA in drinking water (MDH 2011).
  - New Jersey has established a preliminary health-based guidance value of 0.04 µg/L for PFOA in drinking water (NJDEP 2013).
  - North Carolina has established an interim maximum allowable concentration (IMAC) of 2 µg/L for PFOA in groundwater (NCDENR 2006).
    - In 2010, the North Carolina Secretary's Science Advisory Board (NCSAB) on Toxic Air Pollutants recommended that the IMAC be reduced to 1 µg/L based on a review of the toxicological literature and discussions with scientists conducting research on the health effects associated with exposure to PFOA. As of February 2014, the NCSAB's recommendation was still pending review by the North Carolina Division of Water Quality (NCSAB 2010).
- Under the Toxic Substances Control Act (TSCA), the EPA finalized two SNURs in 2002 for 88 PFOS-related substances, which require companies to notify the EPA 90 days before starting to manufacture or importing these substances for a significant new use; this pre-notification allows time to evaluate the new use (EPA 2002a, 2013a).
- In 2007, the SNURs were amended to include 183 additional PFOS-related substances (EPA 2006a, 2013a).

Are there any federal and state guidelines and health standards for PFOS and PFOA? (continued)

- On September 30, 2013, the EPA issued a final SNUR requiring companies to report 90 days in advance of all new uses of long-chain perfluoroalkyl carboxylic (LCPFAC) chemicals (defined as having perfluorinated carbon chain lengths equal to or greater than seven carbons and less than or equal to 20 carbons) for use as part of carpets or to treat carpets, including the import of new carpet containing LCPFACs. In addition, the EPA is amending the existing SNUR to add PFOS-related substances that have completed the TSCA new chemical review process but have not yet commenced production or importation, and to designate processing as a significant new use (EPA 2012, 2013a).
- The SNURs allow for continued use for a few highly technical applications of PFOS-related substances where no alternatives are available; these specialized uses are characterized by very low volume, low exposure and low releases (EPA 2009c, 2013a).
- The Agency for Toxic Substances and Disease Registry has not established a minimal risk level (MRL) for PFOS or PFOA; when the draft toxicological profile was published, human studies were insufficient to determine with a sufficient degree of certainty that the effects are either exposure-related or adverse (ATSDR 2009).
- The EPA has not derived a chronic oral reference dose (RfD) or chronic inhalation reference concentration (RfC) for PFOS or PFOA and has not classified PFOS or PFOA carcinogenicity.
- The EPA removed PFOS and PFOA from the Integrated Risk Information System (IRIS) agenda in a Federal Register notice released on October 18, 2010. At this time, EPA is not conducting an IRIS assessment for these chemicals (EPA 2010).
- PFOS and PFOA were included on the third drinking water contaminant candidate list, which is a list of unregulated contaminants that are known to, or anticipated to, occur in public water systems and may require regulation under the Safe Drinking Water Act (EPA 2009a).

What detection and site characterization methods are available for PFOS and PFOA?

- PFOS and PFOA are commonly deposited in the environment as discrete particles with strongly heterogeneous spatial distributions. Unless precautions are taken, this distribution causes highly variable soil data that can lead to confusing or contradictory conclusions about the location and degree of contamination. Proper sample collection (using an incremental field sampling approach), sample processing (which includes grinding) and incremental subsampling are required to obtain reliable soil data (EPA 2003, 2013c).
- PFOS and PFOA in anionic form can be extracted from environmental media by conventional methods using either acidification or ion pairing to obtain a neutral form of the analyte. Sample preparation methods used for PFCs have included solvent extraction, ion-pair extraction, solid-phase extraction and column-switching extraction (Flaherty and others 2005).
- Precursors and intermediate degradation products can be extracted using solvents (Dasu and others 2012; Ellington and others 2009).
- Air samples may be collected using high-volume air samplers that employ sampling modules containing glass-fiber filters and glass columns with a polyurethane foam (Jahnke and others 2007a).
- Detection methods for PFCs are primarily based on high-performance liquid chromatography (HPLC) coupled with tandem mass spectrometry (MS/MS). HPLC-MS/MS has allowed for more sensitive determinations of individual PFOS and PFOA in air, water and soil (EFSA 2008; Jahnke and others 2007b; Washington and others 2008).
- Both liquid chromatography (LC)-MS/MS and gas chromatography-mass spectrometry (GC-MS) can be used to identify the precursors of PFOS and PFOA (EFSA 2008).
- EPA Method 537, Version 1.1, is an LC-MS/MS method used to analyze selected perfluorinated alkyl acids in drinking water. While most sampling protocols for organic compounds require sample collection in glass, this method requires plastic sample bottles because PFCs are known to adhere to glass (EPA 2009b).
- The development of LC - electrospray ionization (ESI) MS and LC-MS/MS has improved the analysis of PFOS and PFOA (EFSA 2008).
- Reported sensitivities for the available detection methods include low picograms per cubic meter (pg/m^3) levels in air, high picograms per liter (pg/L) to low ng/L levels in water and high picogram per gram to low ng/g levels in soil (ATSDR 2009).

What technologies are being used to treat PFOS and PFOA?

- Because of their unique physicochemical properties (strong fluorine-carbon bond and low vapor pressure), PFOS and PFOA resist most conventional in situ treatment technologies, such as direct oxidation (Hartten 2009; Vectis and others 2009).
- Factors to consider when selecting a treatment method in all media include: (1) initial concentration of PFCs; (2) the background organic and metal concentration; (3) available degradation time; and (4) other site-specific conditions (Vectis and others 2009).
- Ex situ treatments including activated carbon filters, nanofiltration and reverse osmosis units have been shown to remove PFCs from water; however, incineration of the concentrated waste would be needed for the complete destruction of PFCs (Hartten 2009; MDH 2008; Vectis and others 2009).
- Research into a cost-effective treatment approach for PFOS and PFOA is ongoing (DoD SERDP 2012).
- Alternative technologies studied for PFOS and PFOA degradation in water, soil and solid waste include photochemical oxidation and thermally induced reduction, which have achieved some bench-scale success (Hartten 2009; Vectis and others 2009).
- Laboratory-scale studies have also evaluated sonochemical degradation (that is, ultrasonic irradiation) to treat PFOS and PFOA in groundwater and have reported a sonochemical degradation half-life less than 30 minutes for both PFOS and PFOA (Cheng and others 2008, 2010).
- Results from a laboratory-scale study suggested the promising potential of using a double-layer permeable reactive barrier (DL-PRB) system for the in situ containment of PFC-contaminated soil and groundwater. The DL-PRB system is composed of an oxidant-releasing material layer followed by a layer of quartz sands immobilized with humification enzymes. The system drives enzyme-catalyzed oxidative humification reactions to degrade PFCs in the PRB (DoD SERDP 2013).
- In situ chemical oxidation is being explored as a possible means to treat PFCs in water. Laboratory-scale study results indicate that heat-activated persulfate and permanganate can effectively degrade PFOS and PFOA in water (Liu and others 2012a, b).

Where can I find more information about PFOS and PFOA?

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